The pathogenesis of LAHS is not fully understood but has been associated with structural abnormalities in the inner root sheath.[6] Given a possible role in trichocyte differentiation, it is conceivable that mutations in keratin 14 could contribute to hair disorders such as LAHS. Mutations in the gene for keratin 6HF (keratin 75), found in the medulla and companion layer apposing the inner root sheath, have been identified in some cases of LAHS.[3,7] Hair keratin mutations were not found in other LAHS cases.[7] The authors postulated that another keratin may play a role. Interestingly, epithelial keratins, such as keratin 14, were not evaluated.[7]

The presence of EBS-DM and LAHS in two patients with the same keratin 14 mutation raises the possibility that the common defect contributes to the pathogenesis of both disorders. Better understanding of the pathogenesis of LAHS in the future may help clarify this potential relationship.

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Hair regrowth in a male patient with extensive androgenetic alopecia on estrogen therapy

To the Editor: Androgenetic alopecia (AGA) is a nonscarring pattern hair loss affecting both sexes. There have been no previous reports on very advanced AGA responsive to medical treatment. Here, we present a sex reassignment patient on estrogen therapy who experienced full hair regrowth over completely alopecic scalp.

A 38-year-old Caucasian male-to-female transsexual candidate presented with AGA on August 12, 1988. Physical examination showed alopecia involving the vertex, temporal, parietal, and frontal scalp with good occipital hair density (Table I). Per patient, he developed vellus scalp hair after initiating ethynyl estradiol 0.5 mg daily 17 months ago, subsequently switching to estradiol 0.05 mg transdermal twice weekly 9 months later. Minoxidil 3% solution daily was initiated, then changed to minoxidil 2% solution twice daily and estrone 20 mg/60 mL solution every morning after 2 weeks, resulting in fine terminal hairs of the vertex (Table I and Fig 1, *A*). Estradiol was increased to 0.2 mg transdermal weekly. A hair replacement surgery consult examination on October 5, 1988, showed extensive balding with peripheral "fringe" and few terminal hairs on vertex to occiput, Hamilton grade VI to VII, concluding he was a poor candidate because of extensive baldness.

Aldactone 25 mg twice daily was started May 1990, and increased 3 months later to 100 mg daily, while estrone solution was increased to 30 mg/60 mL twice daily, with progress in further terminal hair growth (Table I and Fig 1, *B*). A follow-up visit on November 9, 1990, showed decreased facial and body hair, with marked increase in permanent scalp hair growth on examination (Table I and Fig 1, *C*). Tretinoin 0.025% gel 3 times weekly was initiated April 1991, with continued excellent scalp hair growth (Table I and Fig 1, *D* and *E*). He underwent sex reassignment surgery in September 1992, and by February 1993, she was

Table I. Summary of in-office visits with accompanying findings and images

Date of visit	Current treatment	Hair growth noted	Associated photograph
August 22, 1988	Estradiol 0.05 mg, Coumadin, Zantac, minoxidil 3%	New patient, "severe" MPB	None
January 4, 1989	Estradiol 0.2 mg, estrone solution, Coumadin, minoxidil 3%	Fine terminal hair regrowth over vertex	Fig 1, *A*
August 6, 1990	Estradiol 0.2 mg, estrone solution, Coumadin, minoxidil 2%, Aldactone 25 mg	Improved regrowth on vertex	Fig 1, *B*
November 9, 1990	Estradiol 0.2 mg, estrone solution, Coumadin, minoxidil 2%, Aldactone 100 mg	Marked increase in scalp hair regrowth	Fig 1, *C*
April 11, 1991	Estradiol 0.2 mg, estrone solution, Coumadin, minoxidil 2%, Aldactone 200 mg	Continued increase in scalp regrowth with hair filling in	Fig 1, *D*
October 14, 1991	Estradiol 0.2 mg, estrone solution, Coumadin, minoxidil 2%, Aldactone 200 mg, tretinoin 0.025%	Almost full regrowth of scalp with 2 in vertex hair length and vertex thinning (vertex and parietal scalp regrowth > frontal regrowth)	Fig 1, *E*

MPB, Male pattern baldness.

taking Aldactone 300 mg daily, medroxyprogesterone 10 mg daily, estradiol 0.05 mg transdermal daily, minoxidil 2% solution twice daily, and estrone 0.5% solution 3 times daily, with self-discontinuation of tretinoin.

She developed some thinning of vertex and frontal hair and, by February 1994, she was on finasteride 5 mg daily—started spring 1993—without significant improvement. Follow-up visits on August 24, 1995; June 10, 1996; and December 1, 1998, showed slight improvement over prior regrowth with new fine vellus hair on parietal scalp and vertex. She was subsequently lost to follow-up.

Finasteride and minoxidil, although not very effective in advanced AGA, have been shown to halt AGA progression and produce mild to moderate regrowth.[1,2] Benefits require indefinite use and are gradually lost upon cessation. Improved efficacy has also been reported when combining minoxidil with finasteride[1] or tretinoin.[2] Spironolactone, an oral antiandrogen, has been shown to halt or mildly improve AGA in women.[3]

Hamilton[4] demonstrated baldness was precluded by pre-adulthood castration, occurred in susceptible subjects after testosterone administration, and was halted without reversal upon testosterone termination. Also, estrogens have been shown to decrease telogen phase duration and increase anagen phase duration in human scalp.[5] Thus, with the demonstration by Garza et al[6] of hair follicle stem cell retention in AGA, we suggest that adding estrogens to antiandrogen therapy—supplemented by bilateral orchiectomy in this case—may revitalize and maintain quiescent AGA hair follicles.

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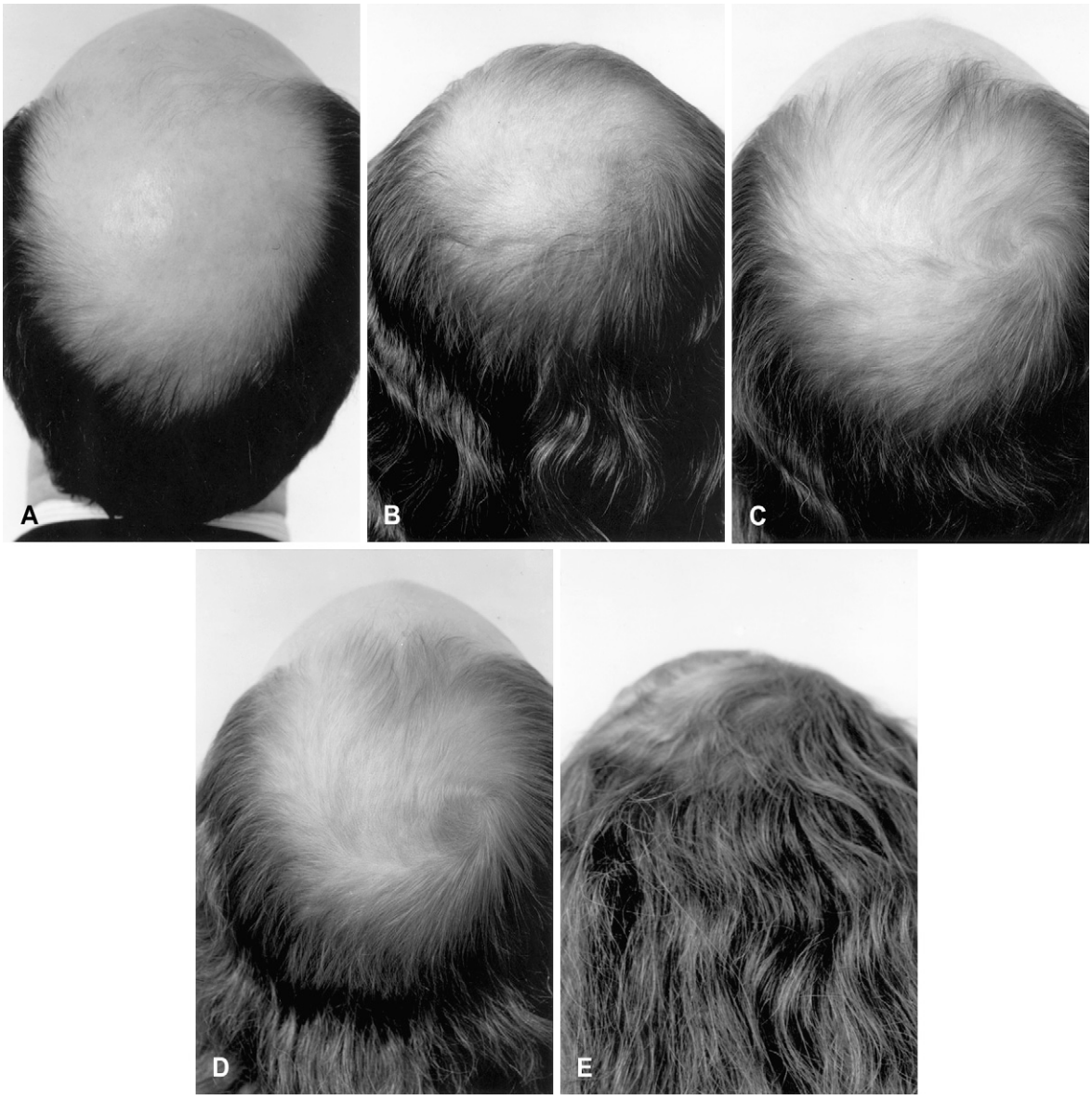

Fig 1. A, Extensive frontal and vertex hair loss. **B**, Improved regrowth of vertex hair. **C**, Continued regrowth of terminal hair on vertex. **D**, Significant regrowth of terminal hair with appreciable coverage on vertex. **E**, Almost full regrowth of terminal hair on scalp with some vertex thinning noted. Photographs taken approximately January 1989 (**A**), August 1990 (**B**), November 1990 (**C**), April 1991 (**D**), and September 1991 (**E**).

Alitretinoin for cutaneous lupus erythematosus

To the Editor: Cutaneous lupus erythematosus (CLE) is an inflammatory autoimmune disease with a broad spectrum of skin manifestations. Thus far, no medication has been approved specifically for the treatment of CLE and only two randomized, placebo-controlled trials are available for discoid lupus erythematosus (DLE) according to the *Cochrane Database of Systematic Reviews*.[1] Antimalarials (ie, hydroxychloroquine) are considered as systemic first-line treatment of CLE, while other medications, such as dapsone and retinoids, are among the second-line treatment options.[2,3] One of the randomized controlled trials suggested that acitretin, an aromatic retinoid, may be as effective as hydroxychloroquine in the treatment of CLE.[4] Another vitamin-A derivative, alitretinoin, has recently been approved for use in severe chronic hand eczema unresponsive to treatment with potent topical corticosteroids.[5]

We report 3 patients with different subtypes of CLE who received off-label systemic treatment